PB88-161948

Health and Environmental Effects Profile for Atrazine

(U.S.) Environmental Protection Agency
Cincinnati, OH

Jan 84

U.S. Department of Commerce
National Technical Information Service

TECHNICAL REPORT DATA
(Please read Instructions on the reverse before completing)

1. REPORT NO. EPA/600/X-84/330	2.
3. RECIPIENT'S ACCESSION NO. PB88 161948/AS	
4. TITLE AND SUBTITLE Health and Environmental Effects Profile for Atrazine	5. REPORT DATE January 1984
	6. PERFORMING ORGANIZATION CODE
7. AUTHOR(S)	8. PERFORMING ORGANIZATION REPORT NO.
9. PERFORMING ORGANIZATION NAME AND ADDRESS	10. PROGRAM ELEMENT NO.
	11. CONTRACT/GRANT NO.
12. SPONSORING AGENCY NAME AND ADDRESS Environmental Criteria and Assessment Office Office of Research and Development U.S. Environmental Protection Agency Cincinnati, OH 45268	13. TYPE OF REPORT AND PERIOD COVERED
	14. SPONSORING AGENCY CODE EPA/600/22
15. SUPPLEMENTARY NOTES	

16. ABSTRACT

The Health and Environmental Effects Profile for atrazine was prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files were evaluated as they pertained to potential human health, aquatic life and environmental effects of hazardous waste constituents.

Quantitative estimates have been presented provided sufficient data are available. Existing data are insufficient to determine an Acceptable Daily Intake (ADI) or a carcinogenic potency factor for atrazine.

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group

18. DISTRIBUTION STATEMENT Public	19. SECURITY CLASS (This Report) Unclassified	21. NO. OF PAGES 70
	20. SECURITY CLASS (This page) Unclassified	22. PRICE A14.95/A6.95 MF

EPA Form 2220-1 (Rev. 4-77) PREVIOUS EDITION IS OBSOLETE

PB88-161948

EPA/600/X-84/330
January 1984

HEALTH AND ENVIRONMENTAL EFFECTS PROFILE FOR ATRAZINE

ENVIRONMENTAL CRITERIA AND ASSESSMENT OFFICE
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PREFACE

Health and Environmental Effects Profiles (HEEPs) are prepared for the Office of Solid Waste by the Office of Health and Environmental Assessment. The HEEPs are intended to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files are evaluated as they pertain to potential human health, aquatic life and environmental effects of hazardous waste constituents. The literature searched and the dates of the searches are included in the section titled "Appendix: Literature Searched."

Quantitative estimates are presented provided sufficient data are available. For systemic toxicants, these include, Acceptable Daily Intakes (ADIs) for chronic exposures. An ADI is defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect. In the case of suspected carcinogens, ADIs are not estimated in this document series. Instead, a carcinogenic potency factor or q_1^* is provided. These potency estimates are derived for both oral and inhalation exposures where possible. In addition, unit risk estimates for air and drinking water are presented based on inhalation and oral data, respectively.

The first draft of this document was prepared by Syracuse Research Corporation under EPA Contract No. 68-03-3228. The document was subsequently revised after reviews by staff within the Office of Health and Environmental Assessment: Carcinogen Assessment Group, Reproductive Effects Assessment Group, Exposure Assessment Group, and the Environmental Criteria and Assessment Office in Cincinnati.

The HEEPs will become part of the EPA RCRA docket.

ACKNOWLEDGEMENTS

The initial draft of this report was prepared by Syracuse Research Corporation under Contract No. 68-03-3228 for U.S. EPA's Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. Dr. Christopher DeRosa was the Technical Project Monitor and Helen Ball was the Project Officer. The final documents in this series were prepared for the Office of Solid Waste, Washington, DC.

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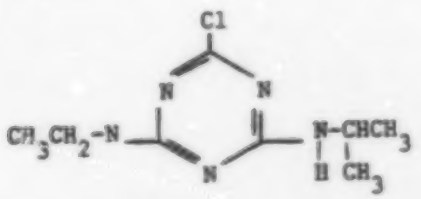
LIST OF ABBREVIATIONS

ADI	Acceptable daily intake
BCF	Bioconcentration factor
GI	Gastrointestinal
GC	Gas chromatography
K_{oc}	Soil adsorption coefficient
LC_{50}	Concentration lethal to 50% of recipients
LD_{50}	Dose lethal to 50% of recipients
NOEL	No-observed-effect level
MS	Mass spectometry
ppb	Parts per billion
ppm	Parts per million
ppmw	Part per million by weight
ppt	Parts per trillion
STEL	Short-term-exposure limit
TLV	Threshold limit value
TWA	Time-weighted average
UV	Ultraviolet

1. INTRODUCTION

1.1. STRUCTURE AND CAS NUMBER

Atrazine is the common name for 2-chloro-4-ethylamino-6-isopropylamino-1,3,5-triazine. Current Chemical Abstracts usage is 6-chloro-N-ethyl-N'-(1-methylethyl)-1,3,5-triazine-2,4-diamine, and was formerly 2-chloro-4-(ethylamino)-6-(isopropylamino)-s-triazine. It was introduced in 1958 as an experimental herbicide by J.R. Geigy S.A. under the code number "G30027" and trademarks "Gesaprim" and "Primatol" (BCPC, 1977; Spencer, 1968). The current Ciba-Geigy trade name for atrazine in the United States is "Aatrex". Other synonyms for atrazine or its formulations include Aatrem, Aktikon, Aktinit, Argezin, Atazinax, Atranex, Atrasine, Atratol, Atred, Candex, Cekuzina-T, Crisazine, Cyazin, Fenamine, Fenatrol, Hungazin, Inakor, Oleogesaprim, Primaze, Radazine, Strazine, Vectal, Weedex A, Wonuk and Zeazine. The structure and empirical formula for atrazine are as follows:



Empirical formula: $C_8H_{14}ClN_5$

Molecular weight: 215.7

The Chemical Abstracts Service (CAS) Registry number for atrazine is 1912-24-9.

1.2. PHYSICAL AND CHEMICAL PROPERTIES

Atrazine is a colorless to white crystalline solid (WSSA, 1979; BCPC, 1977). The technical product is ~98% pure (Spencer, 1968). Selected physical properties are listed below:

Melting point: 173-175°C (WSSA, 1979)

Vapor pressure:

Temperature, °C	mm Hg	
10	5.7×10^{-8}	(WSSA, 1979)
20	3.0×10^{-7}	(WSSA, 1979)
30	1.4×10^{-6}	(WSSA, 1979)
50	2.3×10^{-5}	(WSSA, 1979)
20	2.8×10^{-7}	(Grayson and Fosbraey, 1982)

Solubility:

Solvent	Temperature, °C	ppmw	
dimethyl sulfoxide	27	183,000	(WSSA, 1979)
chloroform	27	52,000	(WSSA, 1979)
ethylacetate	27	28,000	(WSSA, 1979)
methanol	27	18,000	(WSSA, 1979)
diethylether	27	12,000	(WSSA, 1979)
n-pentane	27	360	(WSSA, 1979)
water	27	33	(WSSA, 1979)
water	20	28	(BCPC, 1977)

Log octanol-water partition coefficient:

2.33	(Rao and Davidson, 1982)
2.35	(Rao and Davidson, 1982)
2.71	(Brown and Flagg, 1981)
2.63	(Veith et al., 1979)

Mean soil adsorption coefficient, K_{OC}, from published literature: 163 (Rao and Davidson, 1982)

pK_a 1.62 (20°C) (Plust et al., 1981)

Atrazine is stable in neutral, slightly acidic or basic media, but alkali or mineral acids at higher temperatures hydrolyze it to the hydroxy derivative (BCPC, 1977). It is subject to decomposition by UV irradiation (WSSA, 1979).

1.3. PRODUCTION DATA

Atrazine is prepared by reacting cyanuric chloride with one equivalent of ethylamine at -15 to 0°C and then reacting the product with one equivalent of isopropylamine in the presence of an acid-binding agent (BCPC, 1977; Spencer, 1968).

The following domestic companies are listed as producers of atrazine (SRI, 1983; USITC, 1983a):

Ciba-Geigy Corp.	St. Gabriel, LA
Farmland Industries	St. Joseph, MO
Shell Chemical	Mobile, AL

Ciba-Geigy and Shell Chemical reportedly have plant capacities of 150 and 50 million pounds per year, respectively, to make S-triazine herbicides (SRI, 1976). In 1982, United States production and sales of atrazine were 56.2 and 82.1 million pounds, respectively (USITC, 1983a), while imports of atrazine totalled 0.24 million pounds (USITC, 1983b).

1.4. USE DATA

Atrazine is a widely used selective pre- and post-emergence herbicide that controls broadleaf and grassy weeds in many crops including corn, sorghum, range grass, sugar cane, pineapple, nursery conifers, macadamia orchards and turf grass sod (WSSA, 1979; BCPC, 1977). It is also used widely as a nonselective herbicide for vegetation control in noncrop land. Recommended application rates are 2-4 pounds/acre (WSSA, 1979) or 1-4 kg active ingredient/hectare (BCPC, 1977) for selective weed control, with higher rates used for nonselective control. Atrazine can be used in formulations with other herbicides such as propachlor, sodium chlorate, prometon or metolachlor.

Use of atrazine by American farmers in 1971, 1976 and 1982 was reported to be 53.9, 90.3 and 76.0 million pounds, respectively (USDA, 1983). In 1982, atrazine use in corn was 69.7 million pounds and use in grain sorghum was 6.3 million pounds (USDA, 1983). In 1976, United States farmers used atrazine (in millions of pounds) as follows (Eichers et al., 1978): corn, 83.79; sorghum, 6.53; and wheat, 0.02. Domestic consumption of atrazine (in millions of pounds) in 1975 was reported as follows (SRI, 1976): total, 82.1; corn, 72.4; grain sorghum, 6.7; other field crops, 2.0; and industrial/commercial use, 1.0.

2. ENVIRONMENTAL FATE AND TRANSPORT PROCESSES

2.1. WATER

The degradation of atrazine in water will depend upon its ability to undergo chemical, photochemical and microbial reactions. Its transport in or from water can result from volatilization, sorption onto sediment or suspended matter, and runoff. Each of these mechanisms is discussed below, with the exception of runoff (which is discussed in Section 2.3.), along with bioconcentration and persistence data.

2.1.1. Chemical Hydrolysis. The hydrolysis of atrazine in aqueous conditions has been examined by several authors. Armstrong et al. (1967) found that atrazine hydrolysis in aqueous buffered systems followed first-order kinetics using data collected in pH ranges of 1-4 and 11-13. The hydrolysis half-lives in these ranges were determined as follows:

pH Value	Half-Life in Days (25°C)
1	3.3
2	14
3	58
4	240
11	100
12	12.5
13	1.5

Using a graphical representation from Armstrong et al. (1967), the hydrolysis half-life of atrazine at pH 7 would be ~10,000 days. At pH 3.9, the half-life is just over 200 days; however, the addition of sterilized soil to this pH system was found to increase the hydrolysis rate 10-fold. Therefore, it was demonstrated that atrazine hydrolysis could be significantly catalyzed. The hydrolysis was found to produce hydroxyatrazine as the degradation product.

Li and Felbeck (1972) found that atrazine hydrolysis in a pH range of 1-4 increased with temperature (i.e., at pH 4 and temperatures of 12, 25 and 40°C, the hydrolysis half-lives were 1352, 244 and 49 days, respectively). More significantly, at pH 4, it was found that the addition of a small amount (2%) of humic acid (obtained from soils) as a catalyst dramatically increased the hydrolysis rate. At temperatures of 12, 25 and 40°C, the half-life with humic acid present was 11.34, 1.73 and 0.70 days, respectively. Again, hydroxyatrazine was the degradation product. Khan (1978) studied the catalytic effect of fulvic acid on aqueous hydrolysis of atrazine; fulvic acid occurs naturally in soils and is present in many surface waters. It was found that the hydrolysis rate increased with increasing fulvic acid concentrations and with increasing temperatures. At a fulvic acid concentration of 0.5 mg/mL, the following half-lives at various pH and temperatures were found:

	Half-Life in Days		
pH	25°C	40°C	60°C
2.9	34.8	11.8	3.5
4.5	174	41.7	12.3
6.0	398	95.5	21.9
7.0	742	199	38.9

The only product identified from the hydrolysis was hydroxyatrazine. Perdue (1983) has provided a model that explains atrazine hydrolysis and includes acid-base catalysis and humic acid partitioning equilibria.

Plust et al. (1981) examined the hydrolysis of atrazine at 25 and 39.9°C over a pH range of 0.4-3.1 and found rates similar to those of Armstrong et al. (1967) and Li and Felbeck (1972). In addition, it was found that only acid, base or buffer catalyses of hydrolysis were important; uncatalyzed or water catalyzed reaction does not contribute to hydrolysis (half-life

>267,000 days). Wolfe et al. (1976) also found hydrolysis rates for atrazine to be consistent with those reported by Armstrong et al. (1967). Fernandez-Quintanilla et al. (1981) found that extreme pH values (0.1 or 14.0) completely hydrolyzed atrazine in 3-4 days, while pH values of 1.1 or 13.0 required up to 21 days for complete hydrolysis.

2.1.2. Photodegradation. The UV spectra of atrazine has been charted (Gore et al., 1971; Sadtler Research Laboratories, Inc., 1970) and shows very little absorbency in the environmentally significant range of ≥290 nm. Pape and Zabik (1970) examined the photolysis of atrazine in water, ethanol, methanol and butanol under laboratory conditions. Photolysis did not occur at wavelengths >300 nm. At 253.7 nm, atrazine was photodecomposed to hydroxyatrazine in aqueous solution, while the corresponding alkyloxy analog was formed in alcohol solution. Ruzo et al. (1973) determined rate constants for atrazine photolysis at 300 nm in methanol and butanol solutions; however, the results have little environmental relevance. Wolfe et al. (1976) noted that the direct photolysis of atrazine is so low that it is unlikely to have environmental importance.

The photosensitized photolysis of atrazine has been studied by several authors. Burkhard and Guth (1976) compared the photolysis rates of atrazine in aqueous solution (250 mℓ of 10 mg/ℓ) by itself and with an acetone sensitizer (1 mℓ/100 mℓ) at 15°C and irradiation of ≥290 nm. The degradation was found to be a first-order reaction with a half-life without sensitizer of 25 hours and a half-life with sensitizer of 4.9 hours. After 6 hours of irradiation, the decomposition products were identified as the two de-alkyl-, the de-N,N'-dialkyl- and the hydroxy analogs of atrazine. Tanaka et al. (1981) examined the photolysis of atrazine in aqueous solution (30 ppm) and compared it to the photolysis with addition of 0.2% of two

surface-active agents. The photolyses were carried out at 50°C using a 300 nm sunlamp for a period of 135 minutes. After irradiation, the aqueous solution had lost 8% of the atrazine, while the solutions with the surfactant sensitizers had lost 6 and 17% of the atrazine, respectively. Khan and Schnitzer (1978) found that the photostability of atrazine in fulvic acid was greater than that of an aqueous solution of atrazine without fulvic acid. The photolysis half-life was found to be higher at low rather than at high pH in aqueous fulvic acid. The photolysis of atrazine in water yielded hydroxyatrazine, but additionally yielded three N-dealkylated products in the presence of 0.01% fulvic acid. Rejto et al. (1983) exposed a dye-sensitized solution of atrazine to sunlight for several hours and identified de-ethylated atrazine and 2-chloro-4-(isopropylamino)-6-acetamido-5-triazine and the primary photodegradation products. Kotzias et al. (1982) noted that the photolysis of water plus NO_2^- or NO_3^- at >290 nm yields the OH radical, which can react with atrazine.

2.1.3. Microbial Degradation. The microbial decomposition of atrazine in the soil environment or by soil microorganisms has received considerably more study than its microbial decomposition in the aquatic environment. Data presented in Section 2.3.2. indicate that atrazine can be degraded by microorganisms.

Available data for the aquatic environment suggest that microbial degradation of atrazine is not likely to be the primary mechanism by which it is lost from water. Wolf and Jackson (1982) examined the microbial decomposition of atrazine in a water-sediment system and found that chain-labeled atrazine appeared to be degraded to CO_2 at a higher rate than the ring-labeled material. Geller (1980) compared the degradation of atrazine in sterile solutions via nonbiological mechanisms against solutions to which

bacteria populations from soil, water and sludge were added. The results suggested that atrazine was primarily bound by the bacteria rather than degraded.

2.1.4. Volatilization. Data specific to the volatility of atrazine from aqueous media were not located in the available literature. As noted in Section 2.3., atrazine has some volatility from soil surfaces.

2.1.5. Sorption. Sorption of atrazine onto particulate matter or sediment in water can be an important transport mechanism. In a review of the available data, Wolf and Jackson (1982) concluded that atrazine in the aquatic environment is partitioned, to varying degrees, between an aqueous solution phase and a sediment adsorption phase. Sediments with lower pH and higher total organic carbon levels can be expected to adsorb higher amounts of atrazine than sediments with higher pH and lower organic carbon levels. The conditions of the aquatic environment (pH, organic content, mineral content, temperature) will determine the percentage of atrazine present in solution versus the percentage adsorbed. WSSA (1979) noted that adsorption is not irreversible and desorption can occur depending upon ambient conditions.

2.1.6. Bioconcentration. The bioconcentration of atrazine in aquatic organisms has been studied by a number of authors. Atrazine is judged unlikely to be strongly accumulated in aquatic food chains (Sanborn et al., 1977). In a laboratory stream ecosystem, Lynch et al. (1982) found that the BCFs for crayfish, fish and clams were generally <50. Macek et al. (1976) reported that atrazine failed to accumulate in brook trout, fathead minnows and bluegill sunfish when exposed for 43-44 weeks. It has been reported that in a model terrestrial-aquatic ecosystem, the BCFs for atrazine in alga, snail and fish were only 75.6, 7.5 and 11.0, respectively (Sanborn et

al., 1977). Also, Gunkel and Streit (1980) reported that low levels of atrazine are accumulated in fish and that once the exposed fish are subjected to clean water, the accumulated atrazine is lost rapidly. Ellgehausen et al. (1980) determined BCFs for atrazine in algae, daphnids and catfish as 51, 1.8 and 2.1, respectively, in a simplified model ecosystem. In tests by Streit (1979), atrazine uptake by snails, A. fluviatilis, appeared relatively rapid, with BCFs reaching >300 within 12 hours in water containing a constant atrazine concentration.

Kenaga (1980) predicted BCF values for atrazine, based on the K_{oc} value, and water solubility, to be 7 and 86, respectively; these are consistent with experimental data.

2.1.7. Persistence. Maier-Bode (1972) applied atrazine to a pond and found that it degraded at rates similar to simazine; in 2 weeks, only 10% of the original concentration remained and after 8 weeks, <1.5% remained in the water. After several days, higher concentrations were detected in sediment than in the water and degradation in the sediment was slower than in the water.

Jones et al. (1982) examined the disappearance rates of atrazine from two estuarine water-sediment systems obtained from the Chesapeake Bay and compared the rates to soil persistence in local soils under laboratory conditions. The atrazine half-life in the water was found to be ~3-12 days, while the half-life in the sediment was ~15-20 days. The half-life in the local soils was determined to be ~1 year. The authors also noted that a study by Ballantine et al. (1978) measured a half-life for atrazine of 30 days in Chesapeake Bay sediment.

Weidner (1974) added atrazine to various groundwaters from Nebraska and measured degradation rates under stored laboratory conditions to simulate

groundwater conditions such as lack of light or air effects. The rate of degradation was found to be dependent on concentration, storage temperature, and, to a lesser extent, source of the water. Samples stored at 10°C showed less degradation than samples stored at 25°C. Addition of soil sediment to the water increased degradation. After 15 months of storage, the water only samples still contained up to 80% of the original atrazine.

2.2. AIR

Pertinent data regarding the fate and transport of atrazine in the atmosphere were not located in the available literature. As noted in Section 2.1.2., direct photolysis of atrazine may not be significant, since the UV spectra shows little absorption in the environmentally significant range >290 nm. Photodegradation is more likely in the presence of a photosensitizer.

Cupitt (1980) suggested that adsorption of chemicals on atmospheric aerosols will be a reasonable vapor-phase removal mechanism only for chemicals with saturation vapor pressures of $<10^{-7}$ mm Hg. The vapor pressure of atrazine is 3.0×10^{-7} mm Hg at 20°C and 5.7×10^{-8} mm Hg at 10°C (WSSA, 1979). This indicates the possibility of adsorption at low temperatures.

2.3. SOIL

The fate and transport of atrazine in the soil has been studied more comprehensively than its fate and transport from water or air. The various fate and transport mechanisms are discussed below, along with atrazine's persistence in the soil.

2.3.1. Microbial Degradation. Various researchers have demonstrated that atrazine can be degraded by specific microorganisms. A thorough review of the microbial degradation of S-triazine herbicides was presented by Kaufman and Kearney (1970). Three biodegradation mechanisms have been identified in

atrazine decomposition in the soil: dealkylation, ring cleavage and hydroxylation. Dealkylation is apparently the major mechanism involved in microbial degradation of atrazine. In microbial studies, ^{14}C-chain-labeled atrazine has been shown to evolve $^{14}CO_2$ with the radioactive label tagged at either the ethyl or propyl grouping (Skipper and Volk, 1972). Evolution of $^{14}CO_2$ from microbial systems treated with ^{14}C-ring-labeled atrazine has also been demonstrated; however, only low levels of $^{14}CO_2$ have been evolved, indicating that the S-triazine ring is somewhat resistant to microbial attack (Skipper and Volk, 1972). Under anaerobic conditions, degradation is not extensive [0.02-0.59% mineralization of ring-labeled atrazine (Goswami and Green, 1971)].

While the microbial decomposition of atrazine has been effectively demonstrated, a number of authors have suggested that microbial degradation of atrazine in the soil is not as important as chemical degradation (Leininger, 1970; Skipper et al., 1967; Skipper and Volk, 1972; Geller, 1980; Goswami and Green, 1971).

2.3.2. Chemical Hydrolysis. The chemical hydrolysis of atrazine to the non-phytotoxic hydroxyatrazine has received strong endorsement as a major and, perhaps, predominant mechanism of detoxification of atrazine in soils (Armstrong et al., 1967; Harris, 1967a; Skipper et al., 1967; Skipper and Volk, 1972). Other authors have also suggested that non-biological degradation of atrazine is the major pathway for its disappearance in soils (Zimdahl et al., 1970; Goswami and Green, 1971; Dao et al., 1979; Obien and Green, 1969).

As indicated in Section 2.1.1., the only hydrolysis product identified is 2-hydroxyatrazine. From the model of Perdue (1983), it can be concluded that the rate of hydrolysis in soil will be dependent upon the pH (acid-base catalysis) and the organic content (humic acid catalysis).

2.3.3. Photolysis. The significance of photodecomposition of atrazine from soil is difficult to evaluate, as experiments designed to determine the importance under actual agricultural conditions have not been performed. WSSA (1979) has suggested that loss of atrazine from soil due to photodecomposition has little significance under most field conditions. Photolysis is only likely to occur on the soil surface, as incorporated material is not exposed to irradiation.

As noted in Section 2.1.2., the photosensitized photolysis of atrazine under environmentally significant spectra ranges has been demonstrated in the laboratory. The presence of humic or fulvic acids in soil may sensitize atrazine at the soil surface and permit an unknown degree of photodegradation to occur.

2.3.4. Leaching. Leaching is inversely correlated with adsorption of the herbicide to soil and soil constituents. The relatively large volume of available literature considering atrazine adsorption-leaching indicate that the soil type and conditions can cause significant variances in atrazine's leachability. The measured K_{oc} values in soil of 163 (Rao and Davidson, 1982) and 216 (Brown and Flagg, 1981) suggest only moderate transport in soil.

Helling (1970) and Sanborn et al. (1977) have reviewed atrazine leaching-adsorption and concluded that mobility of atrazine in soils appears to be dependent upon factors that include organic content, pH, temperature, moisture content and mineral content. Soil column and soil TLC studies indicate that atrazine has moderate or intermediate mobility (Rao and Davidson, 1979; Harris, 1967b; Helling and Dragun, 1981; Weber and Whitacre, 1982). Field work indicates that most atrazine stays in the top 15-30 cm (Wu, 1980; Hall and Hartwig, 1978; Agapov and Tulupova, 1980; Tabagua, 1975).

2.3.5. Runoff. Runoff is a transport mechanism that occurs when rainfall rates (or irrigation) exceed the field soil water-infiltration capacity, allowing water to flow over field surfaces and into streams or other locations. Atrazine that has been applied to the soil can be dissolved into the runoff water or be carried by runoff sediment in a sorbed state. A number of researchers have examined atrazine levels in runoff waters from fields to which it has been applied (Ritter et al., 1974; Ellis et al., 1977; Spalding et al., 1979; Hall et al., 1972; Muir et al., 1978; Gaynor and Volk, 1981; Muir and Baker, 1976; Wu, 1980; Triplett et al., 1978; White et al., 1967; Wu et al., 1983; Rohde et al., 1981; Burwell et al., 1974). The loss of atrazine in runoff, as a percentage of atrazine applied, varied in the above studies from <1% to as high as 75%. The runoff loss was influenced by differing farming practices, topography, soil type, drainage characteristics, rainfall intensity and time after atrazine application, as well as other factors. Under normal agricultural conditions and rainfall, however, loss of atrazine due to runoff was generally found to be ~1%.

2.3.6. Volatilization. WSSA (1979) has reported that the significance of atrazine volatilization from soil is not fully understood and that the available data indicate that volatilization from soil surfaces will occur to some extent under favorable environmental conditions.

Foy (1963) found that atrazine deposited on nickel planchets did not evaporate significantly at 25°C under a laboratory hood fan, but exposure of these planchets to infrared lamps at 52-61°C caused 95% of the atrazine to volatilize in 16 hours. Jordan et al. (1970) reported that atrazine loss from metal planchets due to volatilization at room temperature was 2% in 5 hours, 8% in 7 days and 61% in 36 days.

Kearney et al. (1964) found that, depending on the type of soil studied, from 18-32% of the atrazine applied volatilized in 48 hours at 35°C and from 27-52% of the applied atrazine volatilized from the soils at 45°C. Also, atrazine was somewhat less volatile from dry soil than from wet soil. From nickel planchets, Kearney et al. (1964) reported an 80% loss within 24 hours at 25°C and a 95% loss at 35°C within 24 hours.

Burt (1974) studied the volatility of atrazine from glass, plant and soil surfaces by passing air above the surfaces and collecting the volatilized atrazine on a Florisil column. From the glass surfaces, volatility increased with temperature (20-40°C) and air velocity (0.2-2.0 ℓ/minute). Also, concentration was found to be significant, as 70% of a 1 μg application of atrazine volatilized in 2 days, while 10% of a 1000 μg application volatilized in the same time span. A commercial formulation of atrazine (Aatrex) was found to volatilize ~10% more material than technical-grade atrazine. Dried plant material lost 18-27% of applied atrazine in 48 hours at 40°C and 2 ℓ/minute air velocity. The mean loss of atrazine from 10 soil samples at 40°C for 48 hours and 2 ℓ/minute air velocity was 11% of application.

2.3.7. Persistence. The persistence of atrazine in the soil has been studied by numerous authors (Sirons et al. 1973; Muir and Baker, 1978; Hall and Hartwig, 1978; Wu, 1980; Khan et al., 1981; Rohde et al., 1981). Sheets (1970) has written a review of atrazine herbicide persistence and WSSA (1979) has summarized general persistence data concerning atrazine. The following generalizations can be made regarding atrazine persistence in the soil. Atrazine will persist longer under dry and cold conditions or conditions not conducive to maximum chemical or biological activity. Persistence

increases with soil depth and decomposition is more rapid in the summer than in the winter. The growing of tolerant plants may hasten atrazine disappearance. Granular forms of atrazine persist longer than the wettable powder forms. Most rotational crops can be planted 1 year after applications, except in arid or semi-arid climates.

3. EXPOSURE

3.1. WATER

Atrazine has been detected in many natural waters throughout the United States and Canada. Table 3-1 summarizes many of the available studies in which atrazine was monitored in surface and groundwaters. It appears, as a number of these studies have noted, that the atrazine detected is present as a result of agricultural application and subsequent runoff and/or leaching. Hormann et al. (1979) monitored streams in central Europe that drained croplands using atrazine in 1976-1977, and found concentrations >10 ppt, although concentrations were generally lower than those found in the United States.

A number of researchers have examined atrazine levels in runoff waters from fields to which it has been applied (Ritter et al., 1974; Ellis et al., 1977; Spalding et al., 1979; Hall et al., 1972; Muir et al., 1978; Gaynor and Volk, 1981; Muir and Baker, 1976; Wu, 1980; Triplett et al., 1978; White et al., 1967; Wu et al., 1983; Burwell et al., 1974). The loss of atrazine in runoff, as a percentage of atrazine applied, varied from <1-75%. The level of atrazine in runoff waters was apparently influenced by farming practices, topography, soil type, drainage characteristics, intensity of rainfall and time of rainfall after application, as well as other factors.

Atrazine has been detected in finished drinking water. Setzler (1980) found atrazine levels of 3.3-16.4 ppb in Tiffin, OH, city water. The author noted that the levels were similar to monitored levels in the city water source (Sandusky River), indicating that the water treatment process did little to remove the atrazine. Richard et al. (1975) detected atrazine in finished drinking waters from Iowa cities. Of 11 city waters tested, 8 cities had detectable atrazine levels, with the highest concentrations being

TABLE 3-1

Detection of Atrazine in Various Surface and Groundwaters in the United States and Canada

Monitoring Location	Monitoring Dates	Atrazine Concentrations	Comments	Reference
15 Ohio streams	mid-May to mid-July 1980	0.1-45.7 ppb	High percentage of agricultural acreage in the monitoring area is treated with atrazine. More than 20% of samples tested had atrazine levels >5 ppb.	Setzler, 1980
14 Irrigation wells in Nebraska	Aug. 1, 1978	0.06-3.12 ppb	Water was collected beneath irrigated farmland that frequently used atrazine as a corn herbicide.	Spalding et al., 1980
18 Irrigation wells	Aug. 1978	<0.005-6.96 ppb	Same as above.	Spalding et al., 1979
Pawtuxet River, RI	June 1977 to Sept. 1977	3-1400 ppm	Atrazine was detected in river sediments only. It was not detected in water. Positive sediment detection in 20 samples.	Lopez-Avila and Hites, 1980
Iowa surface waters from major watersheds	April 1974 to Sept. 1974	<0.1-42 ppb	Atrazine levels decreased with time, following application and subsequent rainfalls.	Richard et al., 1975
Mississippi River in New Orleans, LA	Feb. 1979	not quantified	Atrazine detected in sub-surface water column.	Overton et al., 1980
Nebraska groundwater under corn producing areas	1979-1980	0.2-0.8 ppb	Atrazine levels in groundwater wells below treated fields increased by as much as 0.7 ppb. About 0.07% of atrazine applied to fields was found to leach to 1.5 m below the surface by the end of the growing season.	Wehtje et al., 1981
Rhode River, MD	1976-1979	<0.01-2.19 ppb	Higher atrazine concentrations were detected in the microsurface water interface than in subsurface waters. Atrazine commonly used herbicide in the region.	Hu, 1981

TABLE 3-1 (cont.)

Monitoring Location	Monitoring Dates	Atrazine Concentrations	Comments	Reference
11 Agricultural watersheds in Canadian Great Lakes Basin	May 1975 to April 1977	0.0-1.2 ppb (mean conc.)	Atrazine was detected in 9-54% of samples collected from differing watersheds using various pesticides.	Coote et al., 1982
92 Streams in Canada flowing into Great Lakes	July 11-15, 1977	1.0-3.3 ng/g (mean conc.)	Atrazine detected in 77% of all samples analyzed.	Strons et al., 1973
Watershed in south-western Ontario	1973-1975	0-34.7 ppb	Atrazine present in 89% of all creek waters analyzed. 30% of all cropland on watershed were treated with S-triazine herbicides.	Frank et al., 1979a
Grand and Saugeen River Basins in Canada	NR	NR	About 0.6% of field applied atrazine appeared to be detected at river mouths.	Frank, 1981
237 Agricultural wells in Ontario	1969-1978	0-22.4 ppm	Atrazine detected in 50 of the wells tested. Concentrations were generally in the range of 0.1 ppb to 1 ppm.	Frank et al., 1979b
5 Rivers in Quebec	1974-1975	0.01-26.9 ppb	The rivers drain agricultural land. Losses of atrazine via runoff were estimated to be 0.1-2.9% of total applied.	Muir et al., 1978

NR = Not reported

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483 ppt. At DesMoines, the effectiveness of water treatment in removing atrazine was studied. It was found that water treatment, even using activated carbon filtration, was ineffective in removing substantial amounts of atrazine. Keith et al. (1976) detected atrazine in Ottumwa drinking water (0.1 ppb) and in three New Orleans drinking waters (4.9-5.4 ppb).

The U.S. EPA STORET data base reports the analysis of 1544 whole water (water plus sediment) samples for atrazine. The mean concentration was 2.7 ppb, with a range of 0-190 ppb. Another 3554 whole water samples produced a mean concentration of 0.42 ppb, with a range of 0-200 ppb. Atrazine levels in 753 mud samples were found to have a mean concentration of 0.2 mg/kg, with a range of 0-100 mg/kg.

3.2. FOOD

Little data are available regarding monitoring of atrazine levels in foods. Carey et al. (1978) detected atrazine (at a concentration of <0.01 ppm) in only 1 of 99 field corn kernel samples collected in 1971 during the National Soils Monitoring Program. In the following year of the program, Carey et al. (1979) detected no atrazine in corn kernel samples. In the U.S. FDA Total Diet Study of ready-to-eat-foods, atrazine has not been listed as a contaminant in any foods to date; the study began in 1964.

3.3. INHALATION

Pertinent data regarding monitoring of atrazine levels in air were not located in the available literature. Wu (1981) monitored levels of atrazine in rainwater bulk precipitation samples (which includes dry deposition). The samples were collected with steel pans placed on top of a tall building at the Chesapeake Bay Center for Environmental Studies. Sixty-eight bulk samples were collected during the study from 1977-1978. Atrazine was being

used by local farmers during the test period. The highest atrazine concentration found in the rainwater was 2.19 ppb, detected in May of 1977 when atrazine was being sprayed in local fields. Surprisingly, high mean levels of atrazine were detected in rainwater during the winter months; in February of 1977 and 1978, mean concentrations of 50 and 11 ppt atrazine were measured, respectively. The author suggested that the presence of atrazine in winter rainwater indicates that once atrazine is released into the atmosphere, it can remain there for relatively long periods of time and may be carried long distances.

3.4. DERMAL

Pertinent data regarding monitoring of atrazine on the skin of American workers were not located in the available literature.

4. PHARMACOKINETICS

4.1. ABSORPTION

As described more fully in Section 4.3., after atrazine ingestion the urine (Bakke et al., 1972a; Erickson et al., 1979) and carcass (Baker et al., 1972a) of treated animals contained atrazine and its metabolites, indicating that atrazine was absorbed through the GI tract.

4.2. DISTRIBUTION

Fourteen rats, weighing 310-420 g, each received 0.53 mg of ring-labeled ^{14}C-atrazine in ethanol by gavage (Bakke et al., 1972a). The amount of radioactivity in the urine and feces was determined daily. Four rats were sacrificed 2, 4 and 8 days after a single dose of atrazine and the level of radioactivity in the carcass was determined. Two rats were housed in all-glass metabolism cages and the amount of radioactive CO_2 in the expired air, as well as levels of radioactivity in urine, feces and carcass, were determined. Seventy-two hours after a single dose of atrazine, 15.8% of the label remained in the carcass and <0.1% of the label was recovered in the expired CO_2. The authors considered the very low level of expired $^{14}CO_2$ indicative that rats could not metabolize the triazine ring. Bakke et al. (1972a) reported that fat (0.5-0.1 ppm) and muscle (0.6-0.5 ppm) had very low levels of radioactivity from 2-8 days after atrazine exposure, whereas liver (3.5-1.7 ppm), kidney (4.0-1.7 ppm) and digestive tract (5.8-0.9 ppm) had higher levels over the same period of time. The levels of radioactivity found in the brain (1.6-1.1 ppm), heart (2.5-1.4 ppm) and lung (3.0-2.0 ppm) from 2-8 days after oral exposure were intermediate.

4.3. METABOLISM

In rat liver homogenates, atrazine appeared to undergo N-dealkylation in the microsomal fraction and conjugation to glutathione in the soluble fraction (Dauterman and Muecke, 1974). The conjugation of atrazine with glutathione required a chloro group at the 2- position in the ring and was catalyzed by glutathione S-transferase, an enzyme purified from mouse liver (Guddewar and Dauterman, 1979). In chicken liver in vitro, the role of glutathione was limited to enzyme-mediated conjugation (Foster et al., 1979). Atrazine underwent hydrolysis and partial N-dealkylation when incubated with the 105000 g soluble fraction. Some dechlorination also occurred.

4.4. EXCRETION

Within 72 hours after oral exposure to ring-labeled ^{14}C-atrazine (Bakke et al., 1972a) (see Section 4.3.), 20.3% of the label was found in the feces and 65.5% in the urine. Eighty-five to 95% of the radioactive carbon in the urine was recovered within the first 24 hours (Bakke et al., 1972a), and the authors suggested that ammeline and/or 2-chloro-4,6-diamino-s-triazine were the terminal metabolites in the rat. In a dissertation abstract without data, atrazine was reported to increase mercapturic acid excretion (Meydani, 1982).

In another study, pentobarbital-anesthetized Pittman-Moore mini-pigs (2-4 months old, weighing 30-50 pounds) received 0.1 g of commercial atrazine (80W) in ethanol by gavage (Erickson et al., 1979). The urine was collected for 46 hours and analyzed by GC/MS. Atrazine and its metabolites, one of which was identified as diethylatrazine, were present in the urine only for the first 24 hours after oral exposure to the herbicide. Excretion by routes other than urinary were not studied in the Erickson et al. (1979) experiment.

In an abstract, Thacker (1971) reported that more than half of the radio-activity appeared in the urine within 48 hours after sheep and cattle were dosed (route not specified) with ring-labeled atrazine. When residues from plants grown on soil containing ^{14}C-atrazine were fed to one sheep and two rats, however, 100 and 88-93% of the ^{14}C excreted was contained in the feces (Bakke et al., 1972b). No further data were reported in the abstract.

5. EFFECTS

5.1. CARCINOGENICITY

In a study performed at the Bionetics Research Laboratory under the direction of the National Cancer Institute (Innes et al., 1969; BRL, 1968a), 18 male and 18 female B6C3F1 mice and 18 male and 18 female B6AKF1 mice were treated by gavage from day 7-28 of life with 21.5 mg/kg/day of atrazine in 0.5% gelatin. Thereafter, atrazine was incorporated in the food at a level of 82 ppm. The animals were sacrificed at 18 months. Controls consisted of groups of 36 (18 male and 18 female) animals from each strain, some of which received the vehicle only. No significantly increased cancer incidence was reported in either strain of mice after oral administration of atrazine.

In an abstract, Donna et al. (1981) reported that both intraperitoneal injection and subcutaneous application of atrazine (which constituted 25% of the tested pesticide, Fogard S) at 3-day intervals for a total of 39 days (dose not reported) resulted in peritoneal mesotheliomas and malignant lymphomas. No details were reported.

5.2. MUTAGENICITY

Atrazine was not mutagenic in Salmonella typhimurium tester strains TA98, TA100, TA1535 and TA1538 (Lusby et al., 1979) and in Aspergillus nidulans and Saccharomyces cerevisiae (de Bertoldi et al., 1980). Details of this study were not readily available. A water soluble extract from corn grown in atrazine treated soil was not significantly different from the extract of corn grown in soil not treated with atrazine in its ability to induce mutations in Salmonella typhimurium TA100 (Bakshi et al., 1981). In an abstract, Chollet et al. (1982) reported no increase in forward mutations in Schizosaccharomyces pombe ade 6, in the presence or absence of S-9 prepared from mouse liver or maize.

Several abstracts reported atrazine to be mutagenic. Atrazine was positive in one of three mutagenicity tests (point mutation in S. typhimurium and Z. may and gene conversion in S. cerevisiae; (Waters et al., 1982). Feeding of atrazine to Drosophila melanogaster larvae caused a significant increase in X-linked recessive mutations and in the loss of the X or Y chromosome (Murnik and Nash, 1977). Corn plants grown in atrazine treated soil contained a soluble mutagenic factor that induced gene conversion in S. cerevisiae D4 (Plewa and Gentile, 1976). Van't Hof and Schairer (1982) reported that atrazine increased the number of blue to pink phenotypes in the plant Tradescantia. The minimum effective concentrations were 0.045 g/flower pot for "chronic" exposure and 6.9×10^{-5} mol for a 24-hour exposure. In an abstract of a Russian study, atrazine was reported to cause a high frequency of chlorophyll mutations (Morgan et al., 1982).

In a sperm morphology assay, male mice were injected intraperitoneally with atrazine suspended in 0.25 mℓ of corn oil on 5 consecutive days (Osterloh et al., 1983). The doses were 600, 1200 and 2400 mg/kg/day. All the mice receiving 1200 and 2400 mg/kg/day died. Thirty-five days after the first injection, the surviving mice were killed, a sperm count taken, their sperm morphology assessed and their testes weighed. There was no significant difference between the atrazine-treated mice and the controls. Osterloh et al. (1983) concluded that the mouse-sperm morphology assay was not a suitable means to detect testicular toxins.

Chollet et al. (1982) reported a significant increase in pre-implantation, but not post-implantation, loss of embryos during a dominant lethal mutation test in mice. The increase in pre-implantation losses occurred at the fourth week after treatment with either a single intraperitoneal injection of atrazine (6 mg/kg bw) or with 1 ppm atrazine in the drinking water

for 7 weeks. No clastogenic effects were seen in bone marrow cells following either treatment. Ehling (1980) reported that atrazine, administered to mice at 2000 mg/kg bw by an unspecified route, caused 4% chromosome breaks in bone marrow cells, as compared with 0.7% in controls. When tested in vitro, with and without metabolic activation, atrazine (12.5-100 mM) did not induce chromosomal aberrations or sister chromatid exchanges in Chinese hamster bone marrow cells (Sobels et al., 1983).

5.3. TERATOGENICITY

Pregnant C3H, BL6 and AKR mice were treated with 46.4 mg/kg/day of atrazine given by daily subcutaneous injection beginning on day 6 of gestation for all three strains, and ending on day 14 of gestation for the C3H and BL6 mice, and on day 15 of gestation for the AKR mice (BRL, 1968b). The mice were sacrificed on day 19 of gestation. There were six litters in the C3H strain, 13 litters in the BL6 strain and 15 litters in the AKR strain. There was a significant decrease in the number of live fetuses/litter and an increase in fetal mortality in the C3H strain. The opposite effect, an increase in live fetuses/litter and a decrease in fetal mortality, was reported in the BL6 mice. In the AKR mice, the number of implantations/litter as well as fetal mortality increased. In comparison with controls, all of these changes were statistically significant. Atrazine does not appear to cause fetal malformations; however, relative maternal liver weight was increased in all three strains and maternal weight gain was depressed in the BL6 and AKR strains only. Fetal weight was depressed only in the AKR mice.

5.4. OTHER REPRODUCTIVE EFFECTS

When groups of four rats (strain not reported) were fed atrazine throughout gestation beginning on day 1 of pregnancy at levels of 0, 50,

100, 200, 300, 400, 500 or 1000 mg/kg diet, no effects were seen in the number of pups per litter or in the weaning weight per pup (Peters and Cook, 1973). When groups of 4-6 pregnant rats were injected subcutaneously with atrazine dissolved in DMSO at doses of 50, 100, 200, 300 and 2000 mg/kg on days 3, 6 and 9 of gestation, no effect on the number of pups and resorption sites per litter was seen at dose levels of 50, 100 and 200 mg/kg. At 800 mg/kg, however, one of four rats completely resorbed her entire litter. At 2000 mg/kg administered intraperitoneally on days 3, 6 and 9 of pregnancy, all four rats resorbed their entire litters. When atrazine was injected subcutaneously on days 3, 6 and 9 of gestation in a second test at high doses, increased numbers of resorption sites and decreased numbers of pups per litter occurred at the two dose levels tested, 1000 and 2000 mg/kg. A single subcutaneous injection of atrazine on days 3, 6 or 9 of gestion was given to assess the critical stage of gestation. This experiment was difficult to interpret, since pups per litter were decreased (~50%) only in animals receiving 100 mg/kg on day 6 of gestation, while all other groups, including animals receiving 2000 mg/kg, delivered normal numbers of pups (Peters and Cook, 1973).

5.5. SUBCHRONIC AND CHRONIC TOXICITY

Ten and 50 mg/kg/day of atrazine administered orally to rats every day for 6 months caused decreased growth, leukopenia and disturbances in thiamine and riboflavin (Suschetet et al., 1974). In a Russian study (Nezefi, 1971), rats exposed to 20 mg/kg/day of atrazine for 6 months developed morphological and biological changes in the brain, heart, liver, lungs, kidneys, ovaries and endocrine glands, and eventually died from the treatment. The daily threshold dose was reported to be 0.1 mg/kg (Nezefi, 1971). No additional data were provided in these abstracts.

5.6. OTHER RELEVANT DATA

The LD_{50} for acute oral exposure to atrazine is 3080 mg/kg in rats and 1750 mg/kg in mice (Wiswesser, 1976).

When female Sprague-Dawley rats were injected intraperitoneally with 250 mg/kg of atrazine, the level of liver cyclic-AMP and glycogen phosphoralase increased ~3-fold within 4-6 hours. Within the same time period, liver glycogen decreased but peripheral blood glucose levels increased (Messner et al., 1979). After rats were treated with 2 and 150 mg/kg/day for 60 days (route not specified), the sensitivity of the diaphragm to insulin was reported to decrease (Madar and Giurgea, 1981). Treatment of rats with 750 mg/kg of atrazine (route and duration not reported) caused a decrease in liver, kidney and spleen glucose-6-phosphate dehydrogenase, liver, kidney and spleen aldolase (Radovcic et al., 1978) and liver and kidney ceruloplasmin (Wurth et al., 1982); spleen and liver acid phosphatase activity increased (Wurth et al., 1982). In another abstract, 2 or 150 mg/kg of atrazine administered to rats for 60 days was reported to stimulate the immune response to Escherichia coli, as evidenced by evaluation of serum antibody titers and total protein and γ-globulin concentration (Giurgea et al., 1981). Feeding rats 500 mg/kg of atrazine for 32-35 days did not alter their trained response to a computer-generated light stimulus (Meydani et al., 1982). However, high doses of atrazine were reported to have a neurotoxic effect on peripheral unmyelinated fibers in the sciatic nerve after intraperitoneal exposure to atrazine (Castano et al., 1982). No further details were contained in the abstract. The growth of Ehrlich ascites tumor cells was inhibited by 30-40% in BALB/c mice when the mice received intraperitoneal injections of 120-140 mg/kg/day of atrazine for 6-8 days (Walker et al., 1979).

6. AQUATIC TOXICITY

6.1. ACUTE TOXICITY

The acute lethal levels of atrazine in water have been determined for several species of freshwater fish. As summarized in Table 6-1, most fish, including trout, salmon and bluegills, had 48- or 96-hour LC_{50} values ranging from 6.0-15.0 mg/ℓ. The harlequin fish, Rasbora heteromorpha, appeared quite sensitive, with 24- and 48-hour LC_{50} values of 0.55 and 0.50 ppm in static conditions (Alabaster, 1969).

In the crustacean, Daphnia magna, the 48-hour LC_{50} value was 3.6 mg atrazine/ℓ in static conditions (FWPCA, 1968). Atrazine at 1 mg/ℓ caused 30% mortality or loss of equilibrium in brown shrimp exposed for 48 hours (Butler, 1963). Macek et al. (1976) reported that the 48-hour LC_{50}s for D. magna and the scud, Gammarus fasciatus, were 5.7-6.9 mg/ℓ and for the midge, Chironomus tentans, was 0.72 mg atrazine/ℓ.

6.2. CHRONIC EFFECTS

Studies regarding the effects of atrazine on reproductive success, embryo survival and larval growth have been conducted on several species of freshwater fish. Hiltibran (1967) reported that granular atrazine at 10 ppm did not affect the survival of bluegill, Lepomis macrochirus, or green sunfish fry, Lepomis cyanellus, during an 8-day exposure period. Atrazine as a wettable powder at 10 ppm did not affect the survival of lake chub fry, Erimyzon sucetta, during 8 days of exposure, but similarly exposed smallmouth bass fry, Micropterus dolomieui, survived only 3 days. Hiltibran (1967) also reported that the highest concentrations of granular and wettable powder atrazine that caused no deaths among juvenile bluegills during a 12-day exposure were 10 and 5 ppm, respectively. Macek et al. (1976)

TABLE 6-1

Acute Lethal Effects of Atrazine to Freshwater Fish

Species	Duration (hours)	Concentration (mg/ℓ)	Method	Effect	Reference
Rainbow trout, *Salmo gairdneri*	48	12.6	static	LC_{50}	FWPCA, 1968
Brook trout, *Salvelinus fontinalis*	96	6.3	flowthrough	LC_{50}	Macek et al., 1976
Coho Salmon, *Oncorhynchus kisutch*	96	15.0	static	25% mortality	Lorz et al., 1979
Harlequin fish, *Rasbora heteromorpha*	24	0.55	static	LC_{50}	Alabaster, 1969
	48	0.50	static	LC_{50}	Alabaster, 1969
Bluegill sunfish, *Lepomis macrochirus*	96	6.0	static	LC_{50}	Walker, 1964
Bluegill sunfish, *L. macrochirus*	96	15.0	static	LC_{50}	Klaassen and Kadoum, 1979
Bluegill sunfish, *L. macrochirus*	96	8.0	flowthrough	LC_{50}	Macek et al., 1976
Fathead minnow, *Pimephales promelas*	96	15.0	flowthrough	LC_{50}	Macek et al., 1976

reported that spawning, survival and growth of embryo-larval stages of bluegills and fathead minnows were not affected by atrazine at 0.095 and 0.213 mg/ℓ, respectively. In brook trout, parental survival, egg production and egg hatchability were not affected at atrazine concentrations of <0.72 mg/ℓ. Brook trout fry, however, showed stunted growth after exposure at 0.72, 0.45 and 0.24 mg atrazine/ℓ (Macek et al., 1976). In studies by Birge et al. (1979), atrazine at levels from 0.017-50.9 mg/ℓ produced a dose-related decrease in percent hatchability, survival at hatching and survival at 4 days post-hatching in rainbow trout and channel catfish. At ~5.0 mg atrazine/ℓ, survival of larvae of both fish species was 9-19% of controls at 4 days post-hatching.

In a study of the effects of long-term (28 days) atrazine exposure on benthic invertebrates, Streit and Peter (1978) reported that survival and reproductive success were decreased at 1 ppm. Species studied were two freshwater leeches (Annelida; Helobdella stagnalis and Glossiphonia complanata) and a snail (Gastropoda; Ancylus fluviatilis). The effects of atrazine on the growth and reproduction of Daphnia pulex were studied in experiments lasting 28 or 70 days (Schober and Lampert, 1976). Atrazine at 1, 2, 5 and 10 mg/ℓ caused a dose-related reduction in survival, length of adult, number of young per brood and cumulative number of young. Even at 1.0 mg atrazine/ℓ, the cumulative number of young was ~500 per original animal, while controls had nearly 700 young per animal.

6.3. PLANT EFFECTS

In the freshwater algae, Scenedesmus quadricauda (green alga) and Microcystis aeruginosa (blue alga), the toxicity threshold for inhibition of cell multiplication was 0.03 and 0.003 mg atrazine/ℓ, respectively (Bringmann and Kuhn, 1978). In the green alga, Chlorella vulgaris, no

growth occurred at >5 mg atrazine/ℓ, and other levels (0.25, 0.5 and 2.5 mg/ℓ) resulted in marked inhibition of growth during a 7-day study (Veber et al., 1981). Cox (1982) reported that atrazine was effective in inhibiting growth of Chlorella sorokiniana at levels from 0.014-0.1 mg/ℓ.

In marine algal species, Nitzschia sigma and Thalassiosira fluviatilis, photosynthetic rate, chlorophyll content and cell numbers were decreased by 10^{-5} M atrazine (2.2 mg/ℓ) (Plumley and Davis, 1980). In estuarine vascular plants, Potamogeton pectinatus, Vallisneria americana and Zostera marina, atrazine at 0.65 mg/ℓ inhibited photosynthesis (Correll and Wu, 1982). Photosynthesis in Zannichellia palustris was inhibited at 0.075 mg atrazine/ℓ. In V. americana, 0.12 mg atrazine/ℓ for 30 days caused 100% mortality, 0.012 mg/ℓ for 47 days caused 50% mortality and 0.0032 and 0.0013 mg/ℓ had little or no effect when compared with a control group.

6.4. RESIDUE

In tests by Streit (1979), atrazine remained at a constant concentration in water for >3 days. Uptake by snails, A. fluviatilis, appeared relatively rapid, with BCFs reaching >300 within 12 hours.

In the whitefish, Coregonus fera, residues of atrazine were highest in the brain, liver, gall bladder and GI tract, with low levels in the gills and skeletal muscle (Gunkel, 1981).

6.5. OTHER RELEVANT INFORMATION

The pharmacokinetics of atrazine in the whitefish, C. fera, were studied by Gunkel (1981). Uptake from water and contaminated food occurs quite rapidly (reaching equilibrium within 24 hours), with subsequent distribution to organs by blood. At an exposure level of ~0.25 mg/ℓ, atrazine accumulated in tissues at 0.4-1.0 mg/kg. Accumulated atrazine was highest in tissues having high lipid content. Elimination of atrazine from the fish

occurred through the gills, feces and mucoid skin secretions. Accumulated atrazine was rapidly cleared from the body, with only the gall bladder retaining high levels at 3 days after the 24-day exposure period was terminated. Concerning human consumption of contaminated fish, skeletal muscles did not accumulate high levels of atrazine during the exposure period and were rapidly cleared of the pesticide after treatment was terminated.

7. EXISTING GUIDELINES AND STANDARDS

7.1. HUMAN

The U.S. EPA has set tolerances for the combined residues of atrazine and its metabolites, 2-amino-4-chloro-6-ethylamino-s-triazine, 2-amino-4-chloro-6-isopropylamino-s-triazine and 2-chloro-4,6-diamino-s-triazine on raw agricultural commodities (Code of Federal Regulations, 1982). These tolerances range from 0.02 ppm for meats and milk to 0.25-15 ppm for grains, grasses, fodder and forage.

The ACGIH (1983) has recommended a TWA-TLV of 5 mg/m^3. No TWA-STEL was recommended. The recommended TWA-TLV was derived from a long-term feeding study in dogs where 3.75 mg/kg was reported to be the highest NOEL (ACGIH, 1980). No details of this dog study were provided.

7.2. AQUATIC

Guidelines and standards for the protection of aquatic organisms from the toxic effects of atrazine were not located in the available literature.

8. RISK ASSESSMENT

Based on the study of Innes et al. (1969), atrazine does not appear to be carcinogenic. Atrazine did not cause an increased mutation rate in Salmonella typhimurium (TA98, TA100, TA1535 and TA1538), Aspergillus nidulans, Saccharomyces cerevisiae (de Bertoldi et al., 1980) or Schizosaccharomyces pombe (Chollet et al., 1982). However, Waters et al. (1982) reported that atrazine was mutagenic in an unspecified assay. After atrazine was metabolized by corn plants, one study reported that it was mutagenic in yeast (Plewa and Gentile, 1976), but another reported that it was not mutagenic in bacteria (Bakshi et al., 1981). Atrazine has been reported to cause chromosome breaks in bone marrow cells (Ehling, 1980) and to cause no chromosomal damage to bone marrow cells (Sobels et al., 1980). Mutations were reported in Drosophila after atrazine treatment (Murnik and Nash, 1977). Without providing any data, Challot et al. (1982) reported that atrazine increases pre-implantation losses in a dominant lethal assay. In a teratogenicity study on three strains of mice at Bionetics Research Laboratories (BRL, 1968b), atrazine did not cause increased fetal malformation, but had inconsistent effects on the number of live fetuses/litter, which appeared to be strain specific in mice. In a rat reproductive study, an increased resorption rate occurred at doses $\geq$800 mg/kg (Peters and Cook, 1973). Although referred to by the ACGIH (1980) (see Section 7.1.), no subchronic or chronic toxicity studies other than abstracts of foreign studies were located in the published English literature. The data are insufficient for the derivation of an ADI.

Statements concerning available literature in this document refer to published, quotable sources and are in no way meant to imply that confidential data, which are considered Confidential Business Information (CBI), are

available in summary form as a CBI Appendix that can be accessed by individuals with appropriate clearance. Conclusions from the CBI Appendix regarding risk assessment, however, can be presented in this chapter.

The CBI Appendix to this document reviews other animal studies in which the toxic effects of atrazine exposure were investigated; the additional data were also insufficient for the derivation of an ADI or q_1^*.

9. REFERENCES

ACGIH (American Conference of Governmental Industrial Hygienists). 1980. Documentation of the Threshold Limit Values, 4th ed. (Includes Supplemental Documentation, 1981, 1982, 1983). Cincinnati, OH. p. 31(81), 32(81), 31, 32.

ACGIH (American Conference of Governmental Industrial Hygienists). 1983. Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1983-1984. Cincinnati, OH. p. 11.

Agapov, V.I. and M.YU. Tulupova. 1980. Behavior of atrazine in soil. Vestn. Mosk. Univ. Ser. XVII Pochvoved. 0(4): 55-57. (CA 94:998x)

Alabaster, J.S. 1969. Survival of fish in 164 herbicides, insecticides, fungicides, wetting agents and miscellaneous substances. Int. Pestic. Contr. 11(2): 29-35.

Armstrong, D.E., G. Chester and R.F. Harris. 1967. Atrazine hydrolysis in soil. Soil Sci. Soc. Am. Proc. 31: 61-66.

Baggi, G. 1981. Study of the biodegradability of substituted triazine compounds. Ann. Microbiol. Enzimol. 31: 13-17.

Bakke, J.E., J.D. Larson and C.E. Price. 1972a. Metabolism of atrazine and 2-hydroxyatrazine by the rat. J. Agric. Food Chem. 10(3): 602-607.

Bakke, J.E., R.H. Shimabukuro, K.L. Davison and G.L. Lamoureaux. 1972b. Sheep and rat metabolism of the insoluble ^{14}C-residues present in ^{14}C-atrazine-treated sorghum. Chemosphere. 1(1): 21-24.

Bakshi, K.S., D.J. Brusick and D.D. Sumner. 1981. Mutagenic activity of corn plants grown in untreated and atrazine (Aatrex) treated soil. Environ. Mutagen. 3: 302-303.

Ballantine, L.G., L.C. Newby and B.J. Simoneaux. 1978. Fate of atrazine in a marine environment. 4th Int. Congress of Pesticide Chemistry. IUPAC, Zurich, Switzerland. Abstract No. V-528.

BCPC (British Crop Protection Council). 1977. Atrazine. In: Pesticide Manual, 5th ed., H. Martin and C.R. Worthing, Ed. BCPC Publications, Worcestershire, England. p. 23.

Birge, W.J., J.A. Black and D.M. Bruser. 1979. Toxicity of organic chemicals to embryo-larval stages of fish. Thomas Hunt Morgan Sch. Biol. Sci., Lexington, MA. EPA 560/11-79/007. NTIS PB 80-101637.

Bringmann, G. and R. Kuhn. 1978. Testing of substances for their toxicity threshold: Model organisms Microcystis (Diplocystis) aeruginosa and Scenedesmus quadricauda. Mitt. Intl. Verein. Limnol. 21: 275-284.

BRL (Bionetics Research Laboratories). 1968a. Evaluation of Carcinogenic, Teratogenic and Mutagenic Activities of Selected Pesticides and Industrial Chemicals. Vol. I. Carcinogenic Study. Prepared by BRL, Bethesda, MD. NTIS PB-223159.

BRL (Bionetics Research Laboratories). 1968b. Evaluation of Carcinogenic, Teratogenic and Mutagenic Activities of Selected Pesticides and Industrial Chemicals. Vol. II. Teratogenic Study in Mice and Rats. Prepared by BRL, Bethesda, MD. NTIS PB-223160.

Brown, D.S. and E.W. Flagg. 1981. Empirical prediction of organic pollutant sorption in natural sediments. J. Environ. Qual. 10(3): 382-386.

Burkhard, N. and J.A. Guth. 1976. Photodegradation of atrazine, atraton and ametryne in aqueous solution with acetone as a photosensitizer. Pestic. Sci. 7(1): 65-71.

Burt, G.W. 1974. Volatility of atrazine from plant, soil and glass surfaces. J. Environ. Qual. 3(2): 114-117.

Burwell, R.E., G.E. Schuman, R.F. Piest, R.G. Spomer and T.M. McCalla. 1974. Quality of water discharged from two agricultural watersheds in southwestern Iowa. Water Resource Res. 10(2): 359-365.

Butler, P.A. 1963. Commercial fisheries investigations. U.S. Fish and Wildlife Serv. Circ. 167: 11-25.

Carey, A.E., J.A. Gowen, H. Tai, W.G. Mitchell and G.B. Wiersma. 1978. Pesticide residue levels in soils and crops, 1971 -- National Soils Monitoring Program (III). Pestic. Monit. J. 12(3): 117-136.

Carey, A.E., J.A. Gowen, H. Tai, W.G. Mitchell and G.B. Wiersma. 1979. Pesticide residue levels in soils and crops from 37 states, 1972 -- National Soils Monitoring Program (IV). Pestic. Monit. J. 12(4): 209-229.

Castano, P., V.F. Ferrario and L. Vizzotto. 1982. Sciatic nerve fibres in albino rats after atrazine treatment: A morpho-quantitative study. Int. J. Tissue React. 4(4): 269-275.

Chollet, M.C., N. Degraeve, J. Gilot-Delhalle, A. Colizzi, J. Moutschen and M. Moutschen-Dahmen. 1982. Mutagenic efficiency of atrazine with and without metabolic activation. Mutat. Res. 97: 237-238.

Code of Federal Regulations. 1982. Atrazine, tolerances for residues. 40 CFR 180.220.

Comes, R.D. and F.L. Timmons. 1965. Effect of sunlight on the phytotoxicity of some phenylurea and triazine herbicides on a soil surface. Weeds. 13: 81. (Cited in Jordan et al., 1970)

Coote, D.R., E.M. MacDonald, W.T. Dickinson, R.C. Ostry and R. Frank. 1982. Agriculture and water quality in the Canadian Great Lakes Basin: I. Representative agricultural watersheds. J. Environ. Qual. 11(3): 473-481.

Correll, D.L. and T.L. Wu. 1982. Atrazine toxicity to submersed vascular plants in simulated estuarine microcosms. Aquat. Bot. 14(2): 151-158.

Cox, H.W., Jr. 1982. The effect of 2,4-D, atrazine and diuron on algal physiology and population dynamics. Dissertation Abstr. Int. B. 1983. Virginia Polytech. Inst., Blacksburg, VA. 43(12, Pt. 1): 3903-3904.

Cupitt, L.T. 1980. Project Summary -- Fate of Toxic and Hazardous Materials in the Air Environment. Dec. 1980, U.S. EPA, Environ. Sci. Res. Lab., Research Triangle Park, NC. EPA 600/S3-80-084

Dao, T.H., T.L. Lavy and R.C. Sorensen. 1979. Atrazine degradation and residue distribution in soil. Soil Sci. Soc. Am. J. 43(6): 1129-1134.

Dauterman, W.C. and W. Muecke. 1974. In vitro metabolism of atrazine by rat liver. Pestic. Biochem. Physiol. 4(2): 212-219.

deBertoldi, M., M. Griselli, M. Giovannetti and R. Barale. 1980. Mutagenicity of pesticides evaluated by means of gene-conversion in Saccharomyces cerevisiae and in Aspergillus nidulans. Environ. Mutagen. 2(3): 359-370. (CA 94:42343j)

Donna, A., P.G. Betta, F. Gagliardi, G.F. Ghiazza, M. Gallareto and V. Gabutto. 1981. Preliminary experimental contribution to the study of possible carcinogenic activity of two herbicides containing atrazine-simazine and trifuralin as active principles. Pathologica. 73(1027): 707-721. (CA 98:84658e)

Ehling, U.H. 1980. Chemically-induced mutations in mice. Comm. Eur. Communit. EUR 6388: 234-239. (CA 93:63212m)

Eichers, T.R., P.A. Andrilenas and T.W. Anderson. 1978. Farmers' Use of Pesticides in 1976. Agricultural Economic Report No. 418. USDA, Economics, Statistics, and Cooperatives Service, Washington, DC. p. 37.

Ellgehausen, H., J.A. Guth and H.O. Esser. 1980. Factors determining the bioaccumulation potential of pesticides in the individual compartments of aquatic food chains. Ecotoxicol. Environ. Saf. 4(2): 134-157.

Ellis, B.G., A.E. Erickson, A.R. Wolcott, M. Zabik and R. Leavitt. 1977. Pesticide runoff losses from small watersheds in Great Lakes Basin. Michigan State Univ., East Lansing J. 17805: 1-90.

Erickson, M.D., C.W. Frank and D.P. Morgan. 1979. Determination of s-triazine herbicide residues in urine: Studies of excretion and metabolism in swine as a model to human metabolism. J. Agric. Food Chem. 27(4): 743-746.

Fernandez-Quintanilla, C., M.A. Cole and F.W. Slife. 1981. Microbial and chemical degradation of atrazine in solution. Theory Pract. Use Soil Appl. Herbic., Symp. Dep. Cereales, INIA, Madrid. p. 301-308. (CA 97:194580k)

Foster, T.S., S.U. Khan and M.H. Akhtar. 1979. Metabolism of atrazine by the soluble fraction (105000 g) from chicken liver homogenates. J. Agric. Food Chem. 27(2): 300-303.

Fournier, J.C. and G. Catroux. 1980. The utilization of strains of microorganisms in the study of pesticide biodegradability. Chemosphere. 9: 33-38.

Foy, C.L. 1963. Volatility of various herbicides under laboratory conditions. Western Weed Contr. Conf. 1963, p. 81-82. (CA 64:1279h)

Frank, R. 1981. Pesticides and PCB in the Grand and Saugeen River Basins. J. Great Lakes Res. 7(4): 440-454. (CA 96:176129v)

Frank, R., G.J. Sirons, R.L. Thomas and K. McMillan. 1979a. Triazine residues in suspended solids (1974-1976) and water (1977) from the mouths of Canadian streams flowing into the Great Lakes. J. Great Lakes Res. 5(2): 131-138.

Frank, R., G.J. Sirons and B.D. Ripley. 1979b. Herbicide contamination and decontamination of well waters in Ontario, Canada, 1968-1978. Pestic. Monit. J. 13(3): 120-127.

FWPCA (Federal Water Pollution Control Administration). 1968. Water Quality Criteria: Report of the National Technical Advisory Committee to the Secretary of the Interior. USDI, Washington, DC.

Gaynor, J.D. and V.V. Volk. 1981. Runoff losses of atrazine and terbutryne from unlimed and limed soil. Environ. Sci. Technol. 15(4): 440-443.

Geller, A. 1980. Studies on the degradation of atrazine by bacteria communities enriched from various biotopes. Arch. Environ. Contam. Toxicol. 9(3): 289-305.

Giurgea, R., M. Borsa and N. Bucur. 1981. Immunological reactions of Wistar rats to administration of atrazine and prometryn. Arch. Exp. Veterinaermed. 35(6): 811-815. (Ger.) (CA 96: 175712t)

Gore, R.C., R.W. Hannah, S.C. Pattacini and T.J. Porro. 1971. Infrared and ultraviolet spectra of seventy-six pesticides. J. Assoc. Off. Anal. Chem. 54(5): 1040-1047.

Goswami, K.P. and R.E. Green. 1971. Microbial degradation of the herbicide atrazine and its 2-hydroxy analog in submerged soils. Environ. Sci. Technol. 5: 426-429.

Grayson, B.T. and L.A. Fosbraey. 1982. Determination of the vapour pressure of pesticides. Pestic. Sci. 13: 269-278.

Guddewar, M.B. and W.C. Dauterman. 1979. Studies on a glutathione S-transferase preparation from mouse liver which conjugates chloro-s-triazine herbicides. Pestic. Biochem. Physiol. 12(1): 1-9.

Gunkel, G. 1981. Bioaccumulation of an herbicide (atrazine, S-triazine) in the whitefish (Coregonus fera): Uptake and distribution of the residue in fish. Arch. Hydrobiol. Suppl. 59(2-3): 252-287.

Gunkel, G. and S. Streit. 1980. Mechanisms of bioaccumulation of a herbicide (atrazine, s-triazine) in a freshwater mollusk (Ancylus fluviatilis Muell.) and a fish (Coregonus fera Jurine). Water Res. 14(11): 1573-1584.

Hall, J.K. and N.L. Hartwig. 1978. Atrazine mobility in two soils under conventional tillage. J. Environ. Qual. 7(1): 53-68.

Hall, J.K., M. Pawlus and E.R. Higgins. 1972. Losses of atrazine in runoff water and soil sediment. J. Environ. Qual. 1(2): 172-176.

Harris, C.I. 1967a. Movement of herbicides in soil. Weeds. 15: 214-216.

Harris, C.I. 1967b. Fate of 2-chloro-s-triazine herbicides in soils. J. Agric. Food Chem. 15: 157-162.

Helling, C.S. 1970. Movement of s-triazine herbicides in soils. Res. Rev. 32: 175-210.

Helling, C.S. and J. Dragun. 1981. Soil leaching tests for toxic organic chemicals. In: Tests Protocols for Environmental Fate and Movement of Toxicants. Proc. Symp. Assoc. Off. Anal. Chem. 94th Ann. Mtg., Washington, DC. p. 43-88.

Hiltibran, R.C. 1967. Effects of some herbicides on fertilized fish eggs and fry. Trans. Am. Fish. Soc. 96(4): 414-416.

Hormann, W.D., J.C. Tournayre and H. Egli. 1979. Triazine herbicide residues in central European streams. Pestic. Monit. J. 13(3): 128-131.

Innes, J.R.M., B.M. Ulland, M.G. Valerio, et al. 1969. Bioassay of pesticides and industrial chemicals for tumorigenicity in mice: A preliminary note. J. Natl. Cancer Inst. 42: 1101-1114.

Jones, T.W., W.M. Kemp, J.C. Stevenson and J.C. Means. 1982. Degradation of atrazine in estuarine water/sediment systems and soils. J. Environ. Qual. 11(4): 632-638.

Jordan, L.S., W.J. Farmer, J.R. Goodin and B.E. Day. 1970. Nonbiological detoxication of s-triazine herbicides. Res. Rev. 32: 267-286.

Kaufman, D.D. and P.C. Kearney. 1970. Microbial degradation of s-triazine herbicides. Res. Rev. 32: 235-265.

Kearney, P.C., T.J. Sheets and J.W. Smith. 1964. Volatility of seven s-triazines. Weeds. 12: 83. (Cited in Jordan et al., 1970)

Keith, L.H., A.W. Garrison, F.R. Allen, et al. 1976. Identification of organic compounds in drinking water from thirteen U.S. cities. In: Identification and Analysis of Organic Pollutants in Water. Ann Arbor, MI. p. 329-373.

Kenaga, E.E. 1980. Predicted bioconcentration factors and soil sorption coefficients of pesticides and other chemicals. Ecotox. Environ. Saf. 4: 26-38.

Khan, S.U. 1978. Kinetics of hydrolysis of atrazine in aqueous fulvic acid solution. Pestic. Sci. 9: 39-43.

Khan, S.U. and M. Schnitzer. 1978. UV irradiation of atrazine in aqueous fulvic acid solution. J. Environ. Sci. Health. Part B. B13(3): 299-310. (CA 89:174935m)

Khan, S.U., P.B. Marriage and A.S. Hamill. 1981. Effects of atrazine treatment of a corn field using different application methods, times and additives on the persistence of residues in soil and their uptake by oat plants. J. Agric. Food Chem. 29: 216-219.

Klaassen, H.E. and A.M. Kadoum. 1979. Distribution and retention of atrazine and carbofuran in farm pond ecosystems. Arch. Environ. Contam. Toxicol. 8: 345. (Cited in Spehar et al., 1981)

Kotzias, D., H. Parlar and F. Korte. 1982. Photoreactivity of organic chemicals in water in the presence of nitrate and nitrite. Naturwissenschaften. 69(9): 444-445. (Ger.) [CA 97(25)215190a]

Leininger, V.E. 1970. Microbial decomposition of ring and chain labeled atrazine in Chalmers silt loam soil. Diss. Abstr. Int. B. Purdue University, Lafayette, IN. 30(9): 3943-3944.

Li, G.C. and G.T. Felbeck. 1972. Atrazine hydrolysis as catalyzed by humic acids. Soil Sci. 114: 201-208.

Lopez-Avila, V. and R.A. Hites. 1980. Organic compounds in an industrial wastewater. Their transport into sediments. Environ. Sci. Technol. 14(11): 1382-1390.

Lorz, H.W., S.W. Glenn, R.H. Williams, C.M. Kunkel, L.A. Norris and B.R. Loper. 1979. Effects of selected herbicides on smolting of coho salmon. U.S. EPA, ORD. EPA 600/3-79-071. 103 p.

Lusby, A.F., Z. Simmons and P.M. McGuire. 1979. Variation in mutagenicity of s-triazine compounds tested on four Salmonella strains. Environ. Mutagen. 1(3): 287-290. (CA 94:25959y)

Lynch, T.R., H.E. Johnson and W.J. Adams. 1982. The fate of atrazine and a hexachlorobiphenyl isomer in a naturally-derived model stream ecosystem. Environ. Toxicol. Chem. 1: 179-192.

Macek, K.J., K.S. Buxton, S. Santer, S. Gnilka and J.W. Dean. 1976. Chronic Toxicity of Atrazine to Selected Aquatic Invertebrates and Fishes. U.S. EPA, Ecol. Res. Serv., Duluth, MN. 50 p. EPA 600/3-76-047. (Cited in Lorz et al., 1979)

Madar, I. and R. Giurgea. 1981. Effect of atrazine and prometryn on the glucose consumption and insulin sensitivity of the diaphragm of white rats. Stud. Cercet. Biol., Ser. Biol. Anim. 33(2): 121-125. (CA 97:50903v)

Maier-Bode, H. 1972. Retention of herbicides in water, sludge, and fish after application in fish ponds. Schriftenr. Ver. Wasser-, Boden-, Lufthyg., Berlin-Dahlem. 37: 67-75. (Ger.) (CA 79:9528s)

Messner, B., J. Berndt and J. Still. 1979. Increases in rat liver cyclic AMP and glycogen phosphorylase activity caused by the herbicide atrazine. Biochem. Pharmacol. 28(2): 207-210.

Meydani, M. 1982. Effect of dietary methionine on methylmercury and atrazine toxicities. Diss. Abstr. Int. B. Iowa State University, Ames, IA. 43(4): 1029.

Meydani, M., D.L. Hopper and J.N. Hathcock. 1982. Behavioral effects of dietary methionine methyl mercury hydroxide and atrazine. 66th Annual Meeting of the Federation of American Societies for Experimental Biology, New Orleans, LA. April 15-23, 1982. Fed. Proc. 41(4): 1982. Abstr. 3945.

Morgan, V.V., V.F. Logvinenko, Y.U.G. Merezhinskii, T.V. Lapina and N.V. Grigorenko. 1982. Cytogenetic and genetic activity of the herbicides atrazine, simazin, prometrin and linuron. Cytol. Genet. (USSR). 16(1): 42-45. (CA 96:137898a)

Muir, D.C. and B.E. Baker. 1976. Detection of triazine herbicides and their degradation products in tile-drain water from fields under intensive corn (maize) production. J. Agric. Food Chem. 24(1): 122-125.

Muir, D.C.G. and B.E. Baker. 1978. The disappearance and movement of three triazine herbicides and several of their degradation products in soil under field conditions. Weed Res. 18(2): 111-120.

Muir, D.C.G., J.Y. Yoo and B.E. Baker. 1978. Residues of atrazine and N-deethylated atrazine in water from five agricultural watersheds in Quebec. Arch. Environ. Contam. Toxicol. 7: 221-235.

Murnik, M.R. and C.L. Nash. 1977. Mutagenicity of the triazine herbicides atrazine, cyanazine and simazine in Drosophila melanogaster. J. Toxicol. Environ. Health. 3(4): 691-697.

Nepomiluev, V.F. and T.I. Kuzyakina. 1972. Action of atrazine and simazine on the soil fungi, and decomposition of the herbicides in soil. Biol. Nauki. 15(8): 127-131. (Rus.) (CA 78:12585t)

Nezefi, T.A. 1971. Morphological changes in white rats during the prolonged action of atrazine. Zdravookhr. Turkm. 15(3): 9-12. (Rus.) [CA 78(3)10860p]

Obien, S.R. and R.E. Green. 1969. Degradation of atrazine in four Hawaiian soils. Weed Sci. 17: 509-514.

Osterloh, J., G. Letz, S. Pond and C. Becker. 1983. An assessment of the potential testicular toxicity of 10 pesticides using the mouse-sperm morphology assay. Mutat. Res. 116(3-4): 407-415.

Overton, E.B., S.W. Mascarella, J.A. McFall and J.L. Laseter. 1980. Organics in the water column and air-water interface samples of Mississippi River water. Chemosphere. 9(10): 629-633.

Pape, B.E. and M.J. Zabik. 1970. Photochemistry of bioactive compounds -- Photochemistry of selected 2-chloro- and 2-methylthio-4,6-di(alkylamino)-s-triazine herbicides. J. Agric. Food Chem. 18(2): 202-207.

Perdue, E.M. 1983. Association of Organic Pollutants with Humic Substances: Partitioning Equilibria and Hydrolysis Kinetics in Aquatic and Terrestrial Humic Materials. Ann Arbor Science, Ann Arbor, MI.

Peters, J.W. and R.W. Cook. 1973. Effects of atrazine on reproduction in rats. Bull. Environ. Contam. Toxicol. 9(5): 301-304.

Plewa, M.J. and J.M. Gentile. 1976. Mutagenicity of atrazine: A maize-microbe bioassay. Mutat. Res. 38(4): 287-292.

Plumley, F.G. and D.E. Davis. 1980. The effects of a photosynthesis inhibitor atrazine, on salt marsh edaphic algae, in culture, microecosystems, and in the field. Estuaries. 3(4): 271-277. (CA 94:133648)

Plust, S.J., J.R. Loehe, F.J. Feher, J.H. Benedict and H.F. Herbrandson. 1981. Kinetics and mechanism of hydrolysis of chloro-1,3,5-triazines. Atrazine. J. Org. Chem. 46(18): 3661-3665.

Radovcic, M., B. Straus and V. Stankovic. 1978. Effects of atrazine on glucose-6-phosphate dehydrogenase and aldolase in rat organs. Acta Pharm. Jugosl. 28(3-4): 127-130. (CA 90:162943v)

Rao, P.S.C. and J.M. Davidson. 1979. Adsorption and movement of selected pesticides at high concentrations in soils. Water Res. 13: 375-180.

Rao, P.S.C. and J.M. Davidson. 1982. Rentention and Transformation of Selected Pesticides and Phosphorus in Soil-Water Systems: A Critical Review. U.S. EPA, Athens, GA. EPA 600/3-82-060.

Rejto, M., S. Saltzman, A.J. Acher and L. Muszkat. 1983. Identification of sensitized photooxidation products of s-triazine herbicides in water. J. Agric. Food Chem. 31(1): 138-142.

Richard, J.J., G.A. Junk, M.J. Avery, N.L. Nehring, J.S. Fritz and H.J. Svec. 1975. Analysis of various Iowa waters for selected pesticides: Atrazine, DDE, and dieldrin-1974. Pestic. Monit. J. 9(3): 117-123.

Ritter, W.F., H.P. Johnson, W.G. Lovely and M. Molnau. 1974. Atrazine, propachlor, and diazinon residues on small agricultural watersheds. Runoff losses, persistence, and movement. Environ. Sci. Technol. 8(1): 38-42.

Roberts, G.C., G.J. Sirons, R. Frank and H.E. Collins. 1979. Triazine residues in a watershed in southwestern Ontario (1973-1975). J. Great Lakes Res. 5(3-4): 246-255. (CA 92:105833b)

Rohde, W.A., L.E. Asmussen, E.W. Hauser, M.L. Hester and H.D. Allison. 1981. Atrazine persistence in soil and transport in surface and subsurface runoff from plots in the coastal plain of the southern United States. Agro-Ecosystems. 7(3): 225-238.

Ruzo, L.O., M.J. Zabik and R.D. Schuetz. 1973. Photochemistry of bioactive compounds. Kinetics of selected s-triazines in solution. J. Agric. Food Chem. 21(6): 1047-1049.

Sadtler Research Laboratories, Inc. 1970. 2-Chloro-4-(ethylamino)-6-(isopropylamino)-s-triazine. 16141 UV. Sadtler Research Laboratories, Inc., Philadelphia, PA.

Sanborn, J.R., B.M. Francis and R.L. Metcalf. 1977. The Degradation of Selected Pesticides in Soil: A Review of Published Literature. U.S. EPA, Cincinnati, OH. p. 16-28. EPA 600/9-77-022.

Schober, U. and W. Lampert. 1976. Effects of sublethal concentrations of a herbicide on Daphnia. Naturwissenschaften. 63(5): 241-242.

Setzler, J.V. 1980. Atrazine Residues in Northern Ohio Streams -- 1980. Tech. Rep., Tiffin, Ohio: Water Quality Lab., Heidelberg College.

Sheets, T.J. 1970. Persistence of triazine herbicides in soils. Res. Rev. 32: 287-309.

Sirons, G.J., R. Frank and T. Sawyer. 1973. Residues of atrazine, cyanazine, and their phytotoxic metabolites in a clay loam soil. J. Agric. Food Chem. 21(6): 1016-1020.

Skipper, H.D. and V.V. Volk. 1972. Biological and chemical degradation of atrazine in three Oregon soils. Weed Sci. 20(4): 344-347.

Skipper, H.D., C.M. Gilmour and W.R. Furtick. 1967. Microbial versus chemical degradation of atrazine in soils. Soil Sci. Soc. Am. Proc. 31: 653-656.

Sobels, F.H., A.D. Tates and A.T. Natarajan. 1980. Studies on the induction of chromosome aberrations in mammalian germ cells and somatic cells by chemical mutagens. (Project 3). Environ. Res. Programme. EUR 6388: 306-310. (CA 93:63216r)

Spalding, R.F., M.E. Exner, J.J. Sullivan and P.A. Lyon. 1979. Chemical seepage from a tail water recovery pit to adjacent ground water. J. Environ. Qual. 8(3): 374-383.

Spalding, R.F., G.A. Junk and J.J. Richard. 1980. Pesticides in groundwater beneath irrigated farmland in Nebraska, August 1978. Pestic. Monit. J. 14(2): 70-73.

Spehar, R.L., A.E. Lemke, Q.H. Pickering, T.H. Roush, R.C. Russo and J.D. Yount. 1981. Effects of pollution on freshwater fish. J. Water Pollut. Control Fed. 53(6): 1028-1076.

Spencer, E.Y. 1968. Guide to the Chemicals Used in Crop Protection. Publ. 1093 (5th ed.). Canada Department of Agriculture, Research Branch, Ottawa, Canada. p. 16.

SRI (Stanford Research Institute). 1976. Herbicides. In: Chemical Economics Handbook. Stanford Research Institute, Menlo Park, CA. p. 573.7003K-L, 573.7008I-J.

SRI (Stanford Research Institute). 1983. 1983 Directory of Chemical Producers: United States of America. SRI International, Menlo Park, CA. p. 773.

Streit, B. 1979. Uptake, accumulation, and release of organic pesticides by benthic invertebrates. 3. Distribution of carbon-14-labelled atrazine and carbon-14-labelled lindane in an experimental three-step food chain microcosm. Arch. Hydrobiol. Suppl. 55(3-4): 373-400.

Streit, B. and H.M. Peter. 1978. Long-term effects of atrazine to selected freshwater invertebrates. Arch. Hydrobiol. Suppl. 55(1): 62-77.

Suschetet, M., J. Leclerc, M. Lhuissier and W. Loisel. 1974. Toxicity and nutritional effects, in the rat, of two herbicides. Picloram (4-amino-3,5,6-trichloropicolinic acid) and atrazine (2-chloro-4-ethylamino-6-isopropylamino-S-triazine). Ann. Nutr. Aliment. 28(1): 29-47. (Fre.) (CA 81:146568k)

Tabagua, M.L. 1975. Penetration of herbicides along a profile of reclaimed soils of the Colchis Plain. Kim. Sel'sk. Khoz. 13(1): 64-65. (Rus.) (CA 82:165748f)

Tanaka, F.S., R.G. Wien and E.R. Mansager. 1981. Survey for surfactant effects on the photodegradation of herbicides in aqueous media. J. Agric. Food Chem. 29: 227-230.

Thacker, E.J. 1971. Pesticide metabolism in animals. Int. Symp. Identification Meas. Environ. Pollut., NRC, Ottawa, Ontario. p. 92-97.

Triplett, G.B., Jr., B.J. Conner and W.M. Edwards. 1978. Transport of atrazine and simazine in runoff from conventional and no-tillage corn. J. Environ. Qual. 7(1): 77-84.

USDA (U.S. Department of Agriculture). 1983. Inputs, Outlook and Situation. IOS-2, October, 1983. USDA, Economic Research Service, Washington, DC. p. 6, 9, 10.

USITC (United States International Trade Commission). 1983a. Synthetic Organic Chemicals. U.S. Production and Sales, 1982. USITC Publ. 1422. p. 225, 227.

USITC (United States International Trade Commission). 1983b. Imports of Benzenoid Chemicals and Products 1982. USITC Publ. 1401.

Van't Hof, J. and L.A. Schairer. 1982. Tradescant'a assay system for gaseous mutagens. A report of the U.S. Environmental Protection Agency Gene-Tox Program. Mutat. Res. 99(3): 303-315.

Veber, K., J. Zahradnik, I. Breyl and F. Kredl. 1981. Toxic effect and accumulation of atrazine in algae. Bull. Environ. Contam. Toxicol. 27(6): 872-876.

Veith, G.D., D.L. DeFoe and B.V. Bergstedt. 1979. Measuring and estimating the bioconcentration factor of chemicals in fish. J. Fish Res. Board Canada. 36: 1040-1048.

Voinova, G. and D. Bakalivanov. 1970. Detoxication of certain aminotriazine herbicides by soil bacteria. Med. Fac. Landouwetensch. Rijksuniv. Gent. 35(2): 839-46. (Fre.) (CA 75:117414c)

Walker, C.R. 1964. Simazine and other s-triazine compounds as aquatic herbicides in fish habitants. Weeds. 12(2): 134-139.

Walker, E.M., Jr., G.R. Gale, L.M. Atkins and R.H. Gadsden. 1979. Some effects of atrazine on Ehrlich ascites tumor cells in vitro and in vivo. Bull. Environ. Contam. Toxicol. 22(1-2): 95-102.

Waters, M.D., J.M. Gentile and M.J. Plewa. 1982. An evaluation of the genotoxic properties of pesticides involved in commercial corn production. Environ. Mutagen. 4: 341.

Weber, J.B. and D.M. Whitacre. 1982. Mobility of herbicides in soil columns under saturated- and unsaturated-flow conditions. Weed Sci. 30(6): 579-84.

Wehtje, G., J.R.C. Leavitt, R.F. Spalding, L.N. Mielke and J.S. Schepers. 1981. Atrazine contamination of groundwater in the Platte Valley of Nebraska from nonpoint sources. Sci. Total Environ. 21: 47-51.

Weidner, C.W. 1974. Degradation in groundwater and mobility of herbicides. Off. Water Res. Technol. Washington, DC. 77 p. NTIS PB-239 242/1st.

White, A.W., A.P. Barnett, B.G. Wright and J.H. Holladay. 1967. Atrazine losses from fallow land caused by runoff and erosion. Environ. Sci. Technol. 1: 740-4.

Wiswesser, W.J. 1976. Pesticide Index. The Entomological Society of America, College Park, MD.

Wolf, D.C. and R.L. Jackson. 1982. Atrazine Degradation, Sorption and Bioconcentration in Water Systems. University of Arkansas, Arkansas Water Resources Research Center, Fayetteville, AR. NTIS PB 83-150151.

Wolfe, N.L., R.G. Zepp, G.L. Baughman, K.C. Fincher, and J.A. Gordon. 1976. Chemical and Photochemical Transformation of Selected Pesticides in Aquatic Systems. U.S. EPA, Athens, GA. EPA 600/3-76-067. p. 125-128.

WSSA (Weed Science Society of America). 1979. Atrazine. In: Herbicide Handbook of the Weed Science Society of America, 4th ed. WSSA, Champaign, IL. p. 32-36.

Wu, T.L. 1980. Dissipation of the herbicides atrazine and alachlor in a Maryland corn field. J. Environ. Qual. 9(3): 459-65.

Wu, T.L. 1981. Atrazine residues in estuarine water and the aerial deposition of atrazine into Rhode River, Maryland. Water Air Soil Pollut. 15(2): 173-184. (CA 94: 186704b)

Wu, T.L., D.L. Correll and H.E.H Remenapp. 1983. Herbicide runoff from experimental watersheds. J. Environ. Qual. 12(3): 330-6.

Wurth, G., B. Straus and V. Stankovic. 1982. Effects of atrazine on ceruloplasmin and acid phosphatase in rat liver, kidney and spleen. Acta. Pharm. Jugosl. 32(1): 53-58. (CA 96: 212165q)

Zimdahl, R.L., V.H. Freed, M.L. Montgomery and W.R. Furtick. 1970. The degradation of triazine and uracil herbicides in soil. Weed Res. 10: 18-26.

APPENDIX

LITERATURE SEARCHED

This profile is based on data identified by computerized literature searches of:

CA SEARCH (Files 308, 309, 310, 311, 320)
TOXLINE
RTECS
OHM TADS
STORET
SRC Environmental Fate Data Bases
SANSS
AQUIRE

These searches were conducted in November, 1983. In addition, hand searches were made of Chemical Abstracts (Collective Indices 6 and 7th), and the following secondary sources were reviewed:

ACGIH (American Conference of Governmental Industrial Hygienists). 1980. Documentation of the Threshold Limit Values, 4th ed. (Includes Supplemental Documentation, 1981, 1982, 1983). Cincinnati, OH. 486 p.

ACGIH (American Conference of Governmental Industrial Hygienists). 1983. TLVs: Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1983-1984. Cincinnati, OH. 94 p.

BCPC (British Crop Protection Council). 1977. Pesticide Manual, 5th ed., H. Martin and C.R. Worthing, Ed. British Crop Protection Council. 593 p.

Clayton, G.D. and F.E. Clayton, Ed. 1981. Patty's Industrial Hygiene and Toxicology, 3rd rev. ed., Vol. 2A. John Wiley and Sons, NY. 2878 p.

Clayton, G.D. and F.E. Clayton, Ed. 1981. Patty's Industrial Hygiene and Toxicology, 3rd rev. ed., Vol. 2B. John Wiley and Sons, NY. 2879-3816 p.

Clayton, G.D. and F.E. Clayton, Ed. 1982. Patty's Industrial Hygiene and Toxicology, 3rd rev. ed., Vol. 2C. John Wiley and Sons, NY. 3817-5112 p.

Grayson, M. and D. Eckroth, Ed. 1978-1983. Kirk-Othmer Encyclopedia of Chemical Technology, 3rd ed. John Wiley and Sons, NY. 23 Volumes.

Hamilton, A. and H.L. Hardy. 1974. Industrial Toxicology, 3rd ed. Publishing Sciences Group, Inc., MA. 575 p.

ITII (International Technical Information Institute). 1982. Toxic and Hazardous Industrial Chemicals Safety Manual for Handling and Disposal with Toxicity and Hazard Data. ITII, Tokyo, Japan. 700 p.

NTP (National Toxicology Program). 1983. Carcinogenesis Testing Program. Chemicals on Standard Protocol. Management Status.

Quellette, R.P. and J.A. King. 1977. Chemical Week Pesticide Register. McGraw-Hill Book Co., NY.

Sax, I.N. 1979. Dangerous Properties of Industrial Materials, 5th ed. Van Nostrand Reinhold Co., NY.

SRI (Stanford Research Institute). 1983. Directory of Chemical Producers. Menlo Park, CA.

U.S. EPA. 1982. Chemical Activities Status Report, 3rd ed. (EPACASR). Offices of Pesticides and Toxic Substances, Washington, DC. EPA 560/TIIS-82-002b.

U.S. EPA. 1983. Status Report on Rebuttable Presumption Against Registration (RPAR) or Special Review Process. Registration Standards and the Data Call In Programs. Office of Pesticide Programs, Washington, DC.

U.S. EPA. 1983. CHIB Existing Chemical Assessment Tracking System. Name and CAS Number Ordered Indexes. Office of Toxic Substances, Washington, DC.

USITC (United States International Trade Commission). 1983. Synthetic Organic Chemicals. U.S. Production and Sales, 1982. USITC Publ. 1422. Washington, DC.

Verschueren, K. 1983. Handbook of Environmental Data on Organic Chemicals, 2nd ed. Van Nostrand Reinhold Co., NY.